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 NEWS 17  Dec 17   TOXCENTER enhanced with additional content
 NEWS 18  Dec 17   Adis Clinical Trials Insight now available on STN
 NEWS 19  Jan 29   Simultaneous left and right truncation added to COMPENDEX,
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 NEWS 22  Feb 24   PCTGEN now available on STN
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 NEWS 24  Feb 26   NTIS now allows simultaneous left and right truncation
 NEWS 25  Feb 26   PCTFULL now contains images
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                   structures available in REGISTRY
 NEWS 30  Apr 11   Display formats in DGENE enhanced
 NEWS 31  Apr 14   MEDLINE Reload
 NEWS 32  Apr 17   Polymer searching in REGISTRY enhanced
 NEWS 33  Jun 13   Indexing from 1947 to 1956 added to records in CA/CAPLUS
 NEWS 34  Apr 21   New current-awareness alert (SDI) frequency in
                   WPIDS/WPINDEX/WPIX
 NEWS 35  Apr 28   RDISCLOSURE now available on STN
 NEWS 36  May 05   Pharmacokinetic information and systematic chemical names
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                  added to PHAR
 NEWS 37   May 15  MEDLINE file segment of TOXCENTER reloaded
 NEWS 38   May 15  Supporter information for ENCOMPPAT and ENCOMPLIT updated
 NEWS 39   May 16  CHEMREACT will be removed from STN
 NEWS 40   May 19  Simultaneous left and right truncation added to WSCA
 NEWS 41   May 19  RAPRA enhanced with new search field, simultaneous left and
                   right truncation
 NEWS 42   Jun 06  Simultaneous left and right truncation added to CBNB
 NEWS 43   Jun 06  PASCAL enhanced with additional data
 NEWS 44   Jun 20  2003 edition of the FSTA Thesaurus is now available

 NEWS EXPRESS  April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT
               MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),
               AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
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=> s C3H and coumarate and lignin
L1            8 C3H AND COUMARATE AND LIGNIN

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L1   ANSWER 1 OF 8  AGRICOLA  Compiled and distributed by the National
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ACCESSION NUMBER: 2003:7059 AGRICOLA
DOCUMENT NUMBER: IND23300724
TITLE: Changes in secondary metabolism and deposition of an unusual ***lignin*** in the ref8 mutant of Arabidopsis.
AUTHOR(S): Franke, R.; Hemm, M.R.; Denault, J.W.; Ruegger, M.O.; Humphreys, J.M.; Chapple, C.
AVAILABILITY: DNAL (QK710.P68)
SOURCE: The Plant journal : for cell and molecular biology, Apr 2002. Vol. 30, No. 1. p. 47-59
Publisher: Oxford : Blackwell Sciences Ltd.
ISSN: 0960-7412
NOTE: Includes references
PUB. COUNTRY: England; United Kingdom
DOCUMENT TYPE: Article
FILE SEGMENT: Non-U.S. Imprint other than FAO
LANGUAGE: English

AB The end products of the phenylpropanoid pathway play important roles in plant structure and development as well as in plant defense mechanisms against biotic and abiotic stresses. From a human perspective, phenylpropanoid pathway-derived metabolites influence both human health and the potential utility of plants in agricultural contexts. The last known enzyme of the phenylpropanoid pathway that has not been characterized is p- ***coumarate*** 3-hydroxylase (***C3H***). By screening for plants that fail to accumulate soluble fluorescent phenylpropanoid secondary metabolites, we have identified a number of Arabidopsis mutants that display a reduced epidermal fluorescence (ref) phenotype. We have now shown that the ref8 mutant is defective in the gene encoding ***C3H*** . Phenotypic characterization of the ref8 mutant has revealed that the lack of ***C3H*** activity in the mutant leads to diverse changes in phenylpropanoid metabolism. The ref8 mutant accumulates p- ***coumarate*** esters in place of the sinapoylmalate found in wild-type plants. The mutant also deposits a ***lignin*** formed primarily from p-coumaryl alcohol, a monomer that is at best a minor component in the ***lignin*** of other plants. Finally, the mutant displays developmental defects and is subject to fungal attack, suggesting that phenylpropanoid pathway products downstream of REF8 may be required for normal plant development and disease resistance.

L1 ANSWER 2 OF 8 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved. (2003)

ACCESSION NUMBER: 2003:7058 AGRICOLA
DOCUMENT NUMBER: IND23300722
TITLE: The Arabidopsis REF8 gene encodes the 3-hydroxylase of phenylpropanoid metabolism.
AUTHOR(S): Franke, R.; Humphreys, J.M.; Hemm, M.R.; Denault, J.W.; Ruegger, M.O.; Cusumano, J.C.; Chapple, C.
AVAILABILITY: DNAL (QK710.P68)
SOURCE: The Plant journal : for cell and molecular biology, Apr 2002. Vol. 30, No. 1. p. 33-45
Publisher: Oxford : Blackwell Sciences Ltd.
ISSN: 0960-7412

NOTE: Includes references
PUB. COUNTRY: England; United Kingdom
DOCUMENT TYPE: Article
FILE SEGMENT: Non-U.S. Imprint other than FAO
LANGUAGE: English

AB The activity of p- ***coumarate*** 3-hydroxylase (***C3H***) is thought to be essential for the biosynthesis of ***lignin*** and many other phenylpropanoid pathway products in plants; however, no conditions suitable for the unambiguous assay of the enzyme are known. As a result, all attempts to purify the protein and clone its corresponding gene have failed. By screening for plants that accumulate reduced levels of soluble fluorescent phenylpropanoid secondary metabolites, we have identified a number of Arabidopsis mutants that display a reduced epidermal fluorescence (ref) phenotype. Using radiotracer-feeding experiments, we have determined that the ref8 mutant is unable to synthesize caffeic acid, suggesting that the mutant is defective in a gene required for the activity or expression of ***C3H*** . We have isolated the REF8 gene using positional cloning methods, and have verified that it encodes ***C3H*** by expression of the wild-type gene in yeast. Although many previous reports in the literature have suggested that ***C3H*** is a phenolase, the isolation of the REF8 gene demonstrates that the enzyme is actually a cytochrome P450-dependent monooxygenase. Although the enzyme accepts p- ***coumarate*** as a substrate, it also exhibits significant activity towards other p-hydroxylated substrates. These data may explain the previous difficulties in identifying ***C3H*** activity in plant extracts and they indicate that the currently accepted version of the ***lignin*** biosynthetic pathway is likely to be incorrect.

L1 ANSWER 3 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2003:15437 BIOSIS
DOCUMENT NUMBER: PREV200300015437
TITLE: Trends in ***lignin*** modification: A comprehensive analysis of the effects of genetic manipulations/mutations on lignification and vascular integrity.
AUTHOR(S): Anterola, Aldwin M.; Lewis, Norman G. (1)
CORPORATE SOURCE: (1) Institute of Biological Chemistry, Washington State University, Pullman, WA, 99164-6340, USA: lewisn@wsu.edu USA
SOURCE: Phytochemistry (Amsterdam), (October 2002, 2002) Vol. 61, No. 3, pp. 221-294. print.
ISSN: 0031-9422.
DOCUMENT TYPE: General Review
LANGUAGE: English

AB A comprehensive assessment of ***lignin*** configuration in transgenic and mutant plants is long overdue. This review thus undertook the systematic analysis of trends manifested through genetic and mutational manipulations of the various steps associated with monolignol biosynthesis; this included consideration of the downstream effects on organized ***lignin*** assembly in the various cell types, on vascular function/integrity, and on plant growth and development. As previously noted for dirigent protein (homologs), distinct and sophisticated monolignol forming metabolic networks were operative in various cell types, tissues and organs, and form the cell-specific guaiacyl (G) and guaiacyl-syringyl (G-S) enriched ***lignin*** biopolymers, respectively. Regardless of cell type undergoing lignification, carbon allocation to the different monolignol pools is apparently determined by a combination of phenylalanine availability and cinnamate-4-hydroxylase/"p-

coumarate -3-hydroxylase" (C4H/ ***C3H***) activities, as revealed by transcriptional and metabolic profiling. Downregulation of either phenylalanine ammonia lyase or cinnamate-4-hydroxylase thus predictably results in reduced ***lignin*** levels and impaired vascular integrity, as well as affecting related (phenylpropanoid-dependent) metabolism. Depletion of ***C3H*** activity also results in reduced ***lignin*** deposition, albeit with the latter being derived only from hydroxyphenyl (H) units, due to both the guaiacyl (G) and syringyl (S) pathways being blocked. Apparently the cells affected are unable to compensate for reduced G/S levels by increasing the amounts of H-components. The downstream metabolic networks for G- ***lignin*** enriched formation in both angiosperms and gymnosperms utilize specific cinnamoyl CoA O-methyltransferase (CCOMT), 4- ***coumarate*** :CoA ligase (4CL), cinnamoyl CoA reductase (CCR) and cinnamyl alcohol dehydrogenase (CAD) isoforms: however, these steps neither affect carbon allocation nor H/G designations, this being determined by C4H/ ***C3H*** activities. Such enzymes thus fulfill subsidiary processing roles, with all (except CCOMT) apparently being bifunctional for both H and G substrates. Their severe downregulation does, however, predictably result in impaired monolignol biosynthesis, reduced ***lignin*** deposition/vascular integrity, (upstream) metabolite build-up and/or shunt pathway metabolism. There was no evidence for an alternative acid/ester O-methyltransferase (AEOMT) being involved in ***lignin*** biosynthesis. The G/S ***lignin*** pathway networks are operative in specific cell types in angiosperms and employ two additional biosynthetic steps to afford the corresponding S components, i.e. through introduction of an hydroxyl group at C-5 and its subsequent O-methylation. (These enzymes were originally classified as ferulate-5-hydroxylase (F5H) and caffeate O-methyltransferase (COMT), respectively.) As before, neither step has apparently any role in carbon allocation to the pathway; hence their individual downregulation/manipulation, respectively, gives either a G enriched ***lignin*** or formation of the well-known S-deficient bm3 " ***lignin*** " mutant, with cell walls of impaired vascular integrity. In the latter case, COMT downregulation/mutation apparently results in utilization of the isoelectronic 5-hydroxyconiferyl alcohol species albeit in an unsuccessful attempt to form G-S ***lignin*** proper. However, there is apparently no effect on overall G content, thereby indicating that deposition of both G and S moieties in the G/S ***lignin*** forming cells are kept spatially, and presumably temporally, fully separate. Downregulation/mutation of further downstream steps in the G/S network (i.e. utilizing 4CL, CCR and CAD isoforms) gives predictable effects in terms of their subsidiary processing roles: while severe downregulation of 4CL gave phenotypes with impaired vascular integrity due to reduced monolignol supply, there was no evidence in support of increased growth and/or enhanced cellulose biosynthesis. CCR and CAD downregulation/mutations also established that a depletion in monolignol supply reduced both ***lignin*** contents and vascular integrity, with a concomitant shift towards (upstream) metabolite build-up and/or shunting. The extraordinary claims of involvement of surrogate monomers (2-methoxybenzaldehyde, feruloyl tyramine, vanillic acid, etc.) in lignification were fully disproven and put to rest, with the investigators themselves having largely retracted former claims. Furthermore analysis of the well-known bm1 mutation, a presumed CAD disrupted system, apparently revealed that both G and S ***lignin*** components were reduced. This seems to imply that there is no monolignol specific dehydrogenase, such as the recently described sinapyl alcohol dehydrogenase (SAD) for sinapyl alcohol formation. Nevertheless, different CAD isoforms of differing

homology seem to be operative in different lignifying cell types, thereby giving the G-enriched and G/S-enriched ***lignin*** biopolymers, respectively. For the G- ***lignin*** forming network, however, the CAD isoform is apparently catalytically less efficient with all three monolignols than that additionally associated with the corresponding G/S ***lignin*** forming network(s), which can more efficiently use all three monolignols. However, since CAD does not determine either H, G, or S designation, it again serves in a subsidiary role-albeit using different isoforms for different cell wall developmental and cell wall type responses. The results from this analysis contrasts further with speculations of some early investigators, who had viewed ***lignin*** assembly as resulting from non-specific oxidative coupling of monolignols and subsequent random polymerization. At that time, though, the study of the complex biological (biochemical) process of ***lignin*** assembly had begun without any of the (bio)chemical tools to either address or answer the questions posed as to how its formation might actually occur. Today, by contrast, there is growing recognition of both sophisticated and differential control of monolignol biosynthetic networks in different cell types, which serve to underscore the fact that complexity of assembly need not be confused any further with random formation. Moreover, this analysis revealed another factor which continues to cloud interpretations of ***lignin*** downregulation/mutational analyses, namely the serious technical problems associated with all aspects of ***lignin*** characterization, whether for ***lignin*** quantification, isolation of ***lignin*** -enriched preparations and/or in determining monomeric compositions. For example, in the latter analyses, some 50-90% of the ***lignin*** components still cannot be detected using current methodologies, e.g. by thioacidolysis cleavage and nitrobenzene oxidative cleavage. This deficiency in ***lignin*** characterization thus represents one of the major hurdles remaining in delineating how ***lignin*** assembly (in distinct cell types) and their configuration actually occurs.

L1 ANSWER 4 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:298051 BIOSIS
DOCUMENT NUMBER: PREV200200298051
TITLE: Changes in secondary metabolism and deposition of an unusual ***lignin*** in the ref8 mutant of arabidopsis.
AUTHOR(S): Franke, Rochus; Hemm, Matthew R.; Denault, Jeff W.; Ruegger, Max O.; Humphreys, John M.; Chapple, Clint (1)
CORPORATE SOURCE: (1) Department of Biochemistry, Purdue University, West Lafayette, IN, 47907-1153: chapple@purdue.edu USA
SOURCE: Plant Journal, (April, 2002) Vol. 30, No. 1, pp. 47-59. http://www.blackwell-science.com/cgilib/jnlpage.bin?Journal=TPJ&File=TPJ&Page=aims. print. ISSN: 0960-7412.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The end products of the phenylpropanoid pathway play important roles in plant structure and development, as well as in plant defense mechanisms against biotic and abiotic stresses. From a human perspective, phenylpropanoid pathway-derived metabolites influence both human health and the potential utility of plants in agricultural contexts. The last known enzyme of the phenylpropanoid pathway that has not been characterized is p- ***coumarate*** 3-hydroxylase (***C3H***). By screening for plants that fail to accumulate soluble fluorescent phenylpropanoid secondary metabolites, we have identified a number of

Arabidopsis mutants that display a reduced epidermal fluorescene (ref) phenotype. We have now shown that the ref8 mutant is defective in the gene encoding ***C3H*** . Phenotypic characterization of the ref8 mutant has revealed that the lack of ***C3H*** activity in the mutant leads to diverse changes in phenylpropanoid metabolism. The ref8 mutant accumulates p- ***coumarate*** esters in place of the sinapolymalate found in wild-type plants. The mutant also deposits a ***lignin*** formed primarily from p-coumaryl alcohol, a monomer that is at best a minor component in the ***lignin*** of other plants. Finally, the mutant displays developmental defects and is subject to fungal attack, suggesting that phenylpropanoid pathway products downstream of REF8 may be required for normal plant development and disease resistance.

L1 ANSWER 5 OF 8 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:298050 BIOSIS
DOCUMENT NUMBER: PREV200200298050
TITLE: The Arabidopsis REF8 gene encodes the 3-hydroxylase of phenylpropanoid metabolism.
AUTHOR(S): Franke, Rochus; Humphreys, John M.; Hemm, Matthew R.; Denault, Jeff W.; Ruegger, Max O.; Cusumano, Joanne C.; Chapple, Clint (1)
CORPORATE SOURCE: (1) Department of Biochemistry, Purdue University, West Lafayette, IN, 47907-1153: chapple@purdue.edu USA
SOURCE: Plant Journal, (April, 2002) Vol. 30, No. 1, pp. 33-45. http://www.blackwell-science.com/cgilib/jnlpage.bin?Journal=TPJ&File=TPJ&Page=aims. print. ISSN: 0960-7412.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The activity of p- ***coumarate*** 3-hydroxylase (***C3H***) is thought to be essential for the biosynthesis of ***lignin*** and many other phenylpropanoid pathway products in plants; however, no conditions suitable for the unambiguous assay of the enzyme are known. As a result, all attempts to purify the protein and clone its corresponding gene have failed. By screening for plants that accumulate reduced levels of soluble fluorescent phenylpropanoid secondary metabolites, we have identified a number of Arabidopsis mutants that display a reduced epidermal fluorescence (ref) phenotype. Using radiotracer-feeding experiments, we have determined that the ref8 mutant is unable to synthesize caffeic acid, suggesting that the mutant is defective in a gene required for the activity or expression of ***C3H*** . We have isolated the REF8 gene using positional cloning methods, and have verified that it encodes ***C3H*** by expression of the wild-type gene in yeast. Although many previous reports in the literature have suggested that ***C3H*** is a phenolase, the isolation of the REF8 gene demonstrates that the enzyme is actually a cytochrome P450-dependent monooxygenase. Although the enzyme accepts p- ***coumarate*** as a substrate, it also exhibits significant activity towards other p-hydroxylated substrates. These data may explain the previous difficulties in identifying ***C3H*** activity in plant extracts and they indicate that the currently accepted version of the ***lignin*** biosynthetic pathway is likely to be incorrect.

L1 ANSWER 6 OF 8 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:395298 CAPLUS
DOCUMENT NUMBER: 137:122269
TITLE: Changes in secondary metabolism and deposition of an unusual ***lignin*** in the ref8 mutant of

Arabidopsis

AUTHOR(S): Franke, Rochus; Hemm, Matthew R.; Denault, Jeff W.; Ruegger, Max O.; Humphreys, John M.; Chapple, Clint

CORPORATE SOURCE: Department of Biochemistry, Purdue University, West Lafayette, IN, 47907-1153, USA

SOURCE: Plant Journal (2002), 30(1), 47-59
CODEN: PLJUED; ISSN: 0960-7412

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The end products of the phenylpropanoid pathway play important roles in plant structure and development, as well as in plant defense mechanisms against biotic and abiotic stresses. Phenylpropanoid pathway-derived metabolites influence both human health and the potential utility of plants in agricultural contexts. The last known enzyme of the phenylpropanoid pathway that has not been characterized is p-***coumarate*** 3-hydroxylase (***C3H***). By screening for plants that fail to accumulate sol. fluorescent phenylpropanoid secondary metabolites, a no. of Arabidopsis mutants that display a reduced epidermal fluorescence (ref) phenotype was identified. The ref8 mutant is defective in the gene encoding ***C3H*** . Phenotypic characterization of the ref8 mutant has revealed that the lack of ***C3H*** activity in the mutant leads to diverse changes in phenylpropanoid metab. The ref8 mutant accumulates p- ***coumarate*** esters in place of the sinapolymalate found in wild-type plants. The mutant also deposits a ***lignin*** formed primarily from p-coumaryl alc., a monomer that is at best a minor component in the ***lignin*** of other plants. Finally, the mutant displays developmental defects and is subject to fungal attack, suggesting that phenylpropanoid pathway products downstream of REF8 may be required for normal plant development and disease resistance.

REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 7 OF 8 CAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2002:395296 CAPLUS

DOCUMENT NUMBER: 138:2289

TITLE: The Arabidopsis REF8 gene encodes the 3-hydroxylase of phenylpropanoid metabolism

AUTHOR(S): Franke, Rochus; Humphreys, John M.; Hemm, Matthew R.; Denault, Jeff W.; Ruegger, Max O.; Cusumano, Joanne C.; Chapple, Clint

CORPORATE SOURCE: Department of Biochemistry, Purdue University, West Lafayette, IN, 47907-1153, USA

SOURCE: Plant Journal (2002), 30(1), 33-45
CODEN: PLJUED; ISSN: 0960-7412

PUBLISHER: Blackwell Science Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The activity of p- ***coumarate*** 3-hydroxylase (***C3H***) is thought to be essential for the biosynthesis of ***lignin*** and many other phenylpropanoid pathway products in plants; however, no conditions suitable for the unambiguous assay of the enzyme are known. As a result, all attempts to purify the protein and clone its corresponding gene have failed. By screening for plants that accumulate reduced levels of sol. fluorescent phenylpropanoid secondary metabolites, the authors have identified a no. of Arabidopsis mutants that display a reduced epidermal fluorescence (ref) phenotype. Using radiotracer-feeding expts., the

authors have detd. that the ref8 mutant is unable to synthesize caffeic acid, suggesting that the mutant is defective in a gene required for the activity or expression of ***C3H*** . The authors have isolated the REF8 gene using positional cloning methods, and have verified that it encodes ***C3H*** by expression of the wild-type gene in yeast. Although many previous reports in the literature have suggested that ***C3H*** is a phenolase, the isolation of the REF8 gene demonstrates that the enzyme is actually a cytochrome P 450-dependent monooxygenase. Although the enzyme accepts p- ***coumarate*** as a substrate, it also exhibits significant activity towards other p-hydroxylated substrates. These data may explain the previous difficulties in identifying ***C3H*** activity in plant exts. and they indicate that the currently accepted version of the ***lignin*** biosynthetic pathway is likely to be incorrect.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L1 ANSWER 8 OF 8 CAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2002:142870 CAPLUS
DOCUMENT NUMBER: 136:197192
TITLE: Arabidopsis gene encoding mutant p- ***coumarate*** 3-hydroxylase (***c3h***) and functions in phenylpropanoid metabolism
INVENTOR(S): Chapple, Clinton C. S.; Franke, Rochus; Ruegger, Max O.
PATENT ASSIGNEE(S): Purdue Research Foundation, USA
SOURCE: PCT Int. Appl., 102 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002014497	A2	20020221	WO 2001-US25592	20010816
WO 2002014497	A3	20030313		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2001084952	A5	20020225	AU 2001-84952	20010816
US 2002062496	A1	20020523	US 2001-931267	20010816

PRIORITY APPLN. INFO.: US 2000-225554P P 20000816
WO 2001-US25592 W 20010816

AB The present invention is directed to a method for altering secondary metab. in plants, specifically phenylpropanoid metab. The present invention is further directed to a mutant p- ***coumarate*** 3-hydroxylase (***C3H***) gene, referred to herein as the ref8 gene, its protein product which can be used to prep. gene constructs and transgenic plants. The gene constructs and transgenic plants are further aspects of the present invention. The phenotypic characterization of the

ref8 mutant provided extremely strong evidence that the REF8 gene encodes a protein required for the activity or expression of ***C3H*** . The ref8 mutation affects ***lignin*** biosynthesis qual. The changes in cell wall degradability in the ref8 mutant clearly demonstrate the potential value of down-regulation of ***C3H*** polypeptide activity in plants important to agriculture and forestry. It is clear that similar changes in cell wall characteristics would lead to improved utilization of lignocellulosic material in terms of pulp and paper prodn., and in agricultural process including, but not limited to, the use of forages for animal feedstocks, and the prodn. of other downstream products such as ethanol produced through fermn. processes. The invention also relates to identification of substrate for ***C3H*** . The invention also shows that gene REF8 corresponds to T20B5.9, a gene also annotated as CYP98A3, a putative P 450.

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CA SUBSCRIBER PRICE	0.00	-1.95

STN INTERNATIONAL LOGOFF AT 11:53:21 ON 24 JUN 2003